THE ETIOLOGY OF NONCARIOUS CERVICAL LESIONS: A NEW CHALLENGE FOR THE 21ST CENTURY DENTIST

A ETIOLOGIA DA LESÃO CERVICAL NÃO CARIOSA: UM NOVO DESAFIO PARA O CIRURGIÃO-DENTISTA DO SÉCULO XXI

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Resumo

O termo Lesões Cervicais Não Cariosas (LCNCs) refere-se à perda de tecido dentário duro na Junção Cemento-Esmalte (JCE), cuja etiologia não está relacionada com o envolvimento bacteriano. A origem e a progressão desses defeitos cervicais são consideradas multifatoriais, sendo atribuídas a três fatores principais, sendo eles: abfração, biocorrosão e abrasão. Tais fatores podem estar combinados e associados a eventos de força excessiva aplicada durante a escovação, juntamente com a abrasividade de dentifrícios, hábitos alimentares e/ou parafuncionais. Essas lesões podem ter morfologias diferentes de acordo com seu fator etiológico principal, podendo se apresentar em forma de cunha, oval ou arredondada. O objetivo deste trabalho foi realizar uma revisão de literatura a fim de apresentar os desafios do Cirurgião-Dentista na descoberta dos diferentes fatores etiológicos das LCNCs. O desafio em questão se dá pelo estudo da combinação desses diversos fatores, bem como a realização de um diagnóstico preciso, fazendo-se necessário o conhecimento adequado da etiologia a fim de prevenir futuras novas lesões, estagnar as existentes e assim, capacitar o Cirurgião-Dentista a realizar um tratamento eficaz e longínquo das LCNCs. No entanto, ainda existem muitas controvérsias na literatura, tornando necessária a elaboração de mais estudos para elucidar a etiologia das LCNCs.

Palavras-chave: abrasão dentária, atrito dentário, bruxismo, desgaste dentário, erosão dentária, sensibilidade da dentina.

Abstract

The term noncarious cervical lesions (NCCL) refers to the loss of hard tooth tissue at the Cemento-Enamel Junction (CEJ), whose etiology is not related to bacterial involvement. The origin and progression of these cervical defects are considered multifactorial, being attributed to three main factors: abfraction, biocorrosion and abrasion. Such factors may be combined and associated with excessive force events applied during brushing, along with the abrasiveness of dentifrices, eating and/or parafunctional habits. These lesions may have different morphologies according to their main etiological factor, and may be wedge-shaped, oval or rounded. Our aim in this paper was to perform a literature review in order to present the challenges of the dental surgeon in identifying the different etiological factors of NCCL. Such challenge regards the study of the combination of these factors, as well as the achievement of an accurate diagnosis, requiring adequate knowledge of the etiology in order to prevent future lesions, stagnate existing ones, thus enabling the Dental Surgeon to treat NCCL effectively in the long term. However, there are still many controversies in the literature, making it necessary to develop more studies to elucidate the etiology of NCCL.

Keywords: *tooth wear, tooth abrasion, tooth attrition, tooth erosion, bruxism, and dentin sensitivity.*

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INTRODUCTION

The term noncarious cervical lesions (NCCL) refer to the loss of tooth structure at the cemento-enamel junction (CEJ) without bacterial involvement (1, 2). These lesions can be easily identified on clinical examination, as they present in a very characteristic way as deep or shallow depressions, in disc or wedge-like shapes, located in the cervical region of the teeth (1).

Tooth wear can be considered a physiological factor associated with aging; however, wear becomes pathological when it as a certain degree of severity (3). NCCL have been increasingly frequent in everyday clinical practice (4). Despite some studies show a higher prevalence of these lesions in older individuals (3, 4, 5), currently the frequency of cases in varied age groups has been so constant that such prevalence has become imprecise (4). The different age groups involved, as well as geographic, cultural, socioeconomic factors and higher exposure to some specific risk factors are relevant points for the imprecision of the prevalence of NCCL (4). As for the prevalence according to the location in the dental arch, it is argued that any tooth can present this pathology, although there is a prevalence of certain teeth due to premature contacts and limited saliva protection (6).

The most common consequences of NCCL involve, besides the loss of structural integrity and aesthetic and functional impairment, the occurrence of tooth sensitivity and pulp disorders, which may cause fractures of the affected teeth (1,7). However, the investigation of the causes of this pathology deserves special attention (3).

According to Wood et al. the NCCL seem to have a multifactorial etiology and their shapes are not an accurate guide to identify their cause (7). In the course of time, the NCCL started to be characterized according to a multifactorial pathodynamic mechanism, but there are still disagreements about the causal factors (1). Thus, this has become an area of great professional uncertainty, as it comprises a scope of unclear etiologies and diagnoses that confuses clinical management and arouses disagreement among professionals (7).

In an era of sophisticated dentistry, the risk factors involved in cases of NCCL need to be investigated and exposed before any type of treatment is performed (8). Moreover, there are marked variations in the clinical management decisions of dentists, and the lack of knowledge on the subject is still the main factor related to failure in treating such cases (8). In this context, we aim at presenting the challenges of the dental surgeon to diagnose and treat NCCL in the face of different etiological factors found in the literature.

LITERATURE REVIEW AND DISCUSSION

The researchers conducted a bibliographic survey based on an electronic search in the Pubmed and Scielo databases. The terms used in the search, according to the Medical Subject Headings (MeSH), were: tooth wear, tooth abrasion, tooth attrition, tooth erosion, bruxism, and dentin sensitivity. The search was restricted to articles published between 2004 and 2020, with the exception of four articles: one from 1991, one from 1999, and two from 2000, for historical comparison. In total, we obtained 45 articles and after reading them excluded 12 that were not related to the theme of our paper and/or were not within the planned parameters.

Currently, information is easily disseminated, health promotion policies have gained space and, as a result, a decrease in oral diseases such as caries and periodontal disease has been observed in the daily practice of dentists. However, the incidence of other oral pathologies, such as NCCL, has greatly increased (9, 10).

Despite the high prevalence of this pathology, it has existed for long and mostly in older adults, whose natural dentition presents physiological characteristics of aging or prolonged exposure to factors that cause it (5, 10). Although NCCL have been widely described in the literature, their etiological factors, morphology, and treatment protocols are still being discussed and researched, and current literature states that their etiology is multifactorial (9).

1. Multifactorial etiology

The etiology of NCCL was once attributed exclusively to the abrasiveness of toothbrushes

and toothpastes (11). At other times, some cases were attributed to erosion, caused by acids (12, 13). This denomination (erosion) was later changed to "biocorrosion", due to the forms of chemical, biochemical and electrochemical degradation it encompasses, and the endogenous and exogenous acids, proteolytic agents and piezoelectric effects involved in the phenomenon (14). And the term "abfraction", introduced in the literature by GRIPPO in 1991 became widely used since 2004, designating microfractures in occlusal stress regions of the tooth surface (14, 15, 16). However, in more recent works such as that of 2012, the author considered erroneous the way the term has been interpreted and used, further stating that it has become a trendy term, naming a single etiology to refer all the variables of NCCL (14).

Thus, the appropriate designation of the etiology of NCCL often depends on the combined interaction of these three main factors: abfraction, biocorrosion and abrasion (14). In addition, there are the so-called combination factors, i.e., interacting mechanisms resulting in the etiology of NCCL. Such factors have been exposed by some authors and should be elucidated as follows: salivary buffering capacity; composition, flow rate, pH and viscosity of saliva; tooth composition, shape, structure, mobility, position and maintenance of natural teeth longer in the mouth; prominence and shape of the dental arch; action of the tongue; action of licit and illicit drugs; occupations; mechanical processes resulting from harmful habits; medications and general health problems; enamel and dentin remineralization; food intake; composition and frequency of food and beverage consumption (3, 9, 14). Thus, attributing only one mechanism as the primary or sole cause for NCCL may be inappropriate (14).

2. Abfraction and biomechanics

The phenomenon called abfraction is characterized by the loss of tooth structure in the cervical region of the tooth caused by stress concentration that occurs in this region (9). The constant occlusal biomechanical forces promote the rupture of chemical bonds between enamel and dentin hydroxyapatite crystals, which allows the penetration of small molecules that prevent the reestablishment of bonds, originating microfractures (17, 18). These microfractures form a growing line along the dental neck, which leads to the collapse of the hard tissue, as it makes the enamel layer more fragile and prone to wear (2, 9). The morphology of these lesions presents in an oval shape on its surface and as a wedge in its depth, with sharp internal and external line angles (2, 19, 20).

The prevalence of this type of wear has increased due to several factors such as keeping natural teeth longer in the mouth, new diets rich in acidic substances, habits, environment and lifestyle (3). Some studies point out that the phenomenon of abfraction is related to tissue fatigue that generates enamel fragility over time, which justifies the higher prevalence and severity in older populations, representing the cumulative effects of NCCL (9, 17, 18).

Regarding the prevalence of lesions according to the location of the tooth in the dental arch, it was observed that any tooth can present this pathology. However there is prevalence in premolars, followed by mandibular first molars (5, 6). Incisors and canines can also be affected, but the higher incidence in premolars and lower molars occurs, apparently, due to premature contacts and limited protection of saliva due to the disadvantageous positioning of these teeth in the arch, which may cause little salivary buffering function (6).

Brandini et al. pointed out that most NCCL affect the maxillary premolars on their buccal surfaces, although they can also be located less frequently on the lingual surface (17). Premolars seem to be less able than canines to resist lateral and oblique forces generated by clenching and lateral movement from group function. In this type of disocclusion, contacts among posterior teeth are subjected to a greater force; in addition, due to the position of the premolars in the arch, an inclined force is generated during contact with the antagonists, causing these cusps to incline towards the buccal side during lateral mandibular movements, thus generating cervical stress (17). Soares et al. observed some disadvantages due to the anatomical characteristics of maxillary premolars, associated with higher incidence of fractures in these teeth, such as: presence of furcation in their anatomy, adjacency to the cervical region, presence of marked grooves in

the root and crown and the low coronal volume as compared to molars (21).

Teeth are bodies of complex morphology, therefore it is rare to find only one type of stress (strain, compression or shear); in fact, what usually occurs is a combination of these stresses, resulting in complex stress (18). In addition, excessive occlusal loads can lead to flexure of the tooth, which can give rise to abfracture lesions (7). Thus, the loss of tooth structure is considered a factor of great importance in changing the biomechanical behavior of teeth (22). Therefore, it is possible to consider that the development of NCCL has a relationship with occlusal forces and the mechanisms involved in this process should be understood (17). The biomechanical behavior of teeth undergoes changes when affected by NCCL (22). Such behavior varies according to the direction of the applied occlusal load, generating distribution of stress in the tooth structure, which may be localized or homogeneous (21, 22).

Computer simulation methods such as Finite Element Analysis (FEA) are tools used in a specific computer program to predict the biomechanical behavior of the dental element under loads (axial and oblique), emphasizing the effect of the type of loading in the cervical region of the tooth. It is possible to observe the stretching of enamel, dentin, periodontal ligament and alveolar bone in the form of clear images with numerical stress whose values are represented by a color scale (17, 18, 21).

In the studies by Soares et al, the FEA method allowed the identification and analysis of the point of greatest stress in the tooth structure, revealing that the deepest abfraction lesions are those that concentrate the greatest stresses, determine the severity and influence the evolution of their depth by accelerating the process of tissue loss (21). Machado et al. and Jakupovic et al., who also used the FEA method for stress analysis, observed that axial loading promotes a homogeneous distribution of stresses, whereas oblique loading results in a localized distribution with a high concentration of stress in the cervical region of the tooth on the palatal and buccal surfaces (18, 22), which explains the higher rate of NCCL in teeth more subjected to oblique forces (17).

Brandini et al. pointed out that in addition to enamel fracture lines, occlusal overload also becomes important in the development of NCCL (17). Parafunctional habits and bruxism can generate a diurnal and/or nocturnal dysfunctional activity of the facial muscles that provokes the habit of grinding, clenching and touching the teeth (23, 24). Bruxism is caused by parafunctional mechanical friction that can potentiate the development of NCCL (9). The periodontal ligament seems to be of fundamental importance when in the face of tooth movement imposed by forces generated on the teeth (18). Being composed of collagen fibers that provide high elasticity under load, the periodontal ligament may act as a buffer of stress, absorbing much of the paraxial forces, with protective effect for the emergence and development of NCCL (18).

3. Biocorrosion

According to Amaral et al, the term "erosion" defines the pathological loss of tooth structure caused by a chemical process of acid exposure without bacterial involvement (3). However, Catelan et al. considered more appropriate the term "corrosion", because these lesions originate in corrosive wear and that such term covers biochemical and electrochemical processes involved in this phenomenon, besides the chemical action (25). Since the term "erosion" is related to mechanical degradations and "corrosion" to degradations by chemical effects, Grippo et al. reformulated and named it "biocorrosion" because it is the molecular degradation of the essential properties of a living tissue (14). Currently, both terms, "erosion" and "biocorrosion" are accepted in the dental literature (14, 25).

The morphology of the biocorrosion lesion is presented as defects that are often more rounded, wide, smooth horizontal grooves and prevail towards the lingual side of the teeth. This region is easily exposed to gastric dietary acids from gastroesophageal reflux and is not limited only to the cervical area (2, 19). The intraoral flora does not produce the acids responsible for erosion, as they come from extrinsic or intrinsic sources (25). Factors such as diet (fruits, acidic drinks), environment (chemical industries, chlorinated pools) and medications (vitamin C,

aspirin, and hydrochloric acid) are considered extrinsic causes. As intrinsic causes, we can address the diseases that cause regurgitation of gastric juice or decreased salivary flow (26).

Currently, changes in lifestyle and diet have been fundamental in the search for a healthier life (27). However, new diets rich in acidic foods can be very detrimental to the health and integrity of the tooth structure (9). Acids from the diet are the main cause of tooth erosion due to extrinsic factors (25). Studies by Sobral et al. have shown that the risk of erosion is much higher (specifically, 37 times higher) in people who consume citrus fruits at least twice a day (26). Lesions can also develop with the consumption of apple cider vinegar, at ten times the risk, as well as sports drinks and soft drinks, both at four times the risk if consumed daily (26).

Another extrinsic factor is the acids related to habits, environment and occupations, which come from contamination in chemical industries, metallurgy, steel mills, battery factories in their various physical forms (gases, vapors or mists), constituting an important risk factor responsible for severe and disfiguring dental destruction (3). The profession of enologists (wine tasters) has also been considered an occupation of extreme severity when added to the length of service, the decrease in salivary flow and the buffering capacity of the saliva of each individual, considering that the wine has in its composition acids such as maleic and tartaric, whose pH values are low ranging between pH 2.9 and pH 4.2, bringing with it a highly erosive potential (3, 10). There are also the acids coming from frequent contact with chlorinated pools, as is the case of professional swimmers, water polo players, and divers who are constantly exposed to pool water (27, 29). The big problem is that most pools use high concentrations of chlorine to reduce the proliferation of bacteria, decreasing the pH below the demineralization levels of enamel and dentin, pH 5.5 and pH 6 respectively, causing irreversible loss of tooth structure (27, 29). Added to improper chlorine concentrations, swimmers' base salivary rates are at risk of alteration, further contributing to enamel dissolution (27). Besides swimmers, athletes in general make use of isotonic drinks, which are drinks used with the purpose of rehydration and electrolyte replacement, with highly acidic content at levels below the critical pH for the enamel demineralization (27). As a consequence of dehydration, the low salivary flow reinforces the corrosive process (27).

Individuals who use medications and illicit drugs are also a risk group, because they are associated with a pathological dental wear (3). Amaral et al. defined some: antidepressants, antihypertensives, anticonvulsants, vitamins of continuous use and others responsible for hyposalivation or with low pH as the vitamin C tablets, besides cocaine paste, methamphetamine and lysergic acid (3).

Among the intrinsic causes, gastroesophageal reflux disease (GERD) and regurgitation are the ones commonly responsible for affecting more than half of the population at some point in their lives (3). They were considered the most severe intrinsic destructive factors of enamel and dentin, as they relate to the low pH of gastric juice (3). Soares et al. pointed out GERD as a disorder that causes the displacement of gastric juice from the stomach to the esophagus, manifesting in the oral cavity (27). It is a process caused by episodes of relaxation of the lower esophageal sphincter (27). The most common manifestations are heartburn and regurgitation, and when the latter occurs, dental wear is observed on the palatal and lingual surfaces of the upper and lower teeth (9). Athletes are prone to have reflux pictures due to some exercises that induce the symptoms, such as running and endurance exercises (27). Bulimic patients also have a high rate of dental erosion that may be caused by gastric acid and modified salivary composition at the expense of bulimia (induced and voluntary regurgitation) (25, 28). Another risk group is people over 50 years of age with hiatus hernia. They have a shorter esophagus and thus their stomach is brought into the thoracic region, increasing the likelihood of gastric juice and pancreatic enzymes gaining access to the oral cavity (14).

Grippo et al. pointed to xerostomia, or dry mouth syndrome, as a systemically caused disease that can be both induced by aging and medication use (14). The sensation of dry mouth may be worsened if the individual is a mouth breather due to saliva evaporation. Therefore, xerostomia is a modifying factor of flow rates, buffering capacity, pH and viscosity of

saliva, considering that the latter is responsible for buffering bicarbonates and, consequently, for the scarcity of non-carious lesions in the lingual regions where there is a greater concentration of it (14).

4. Abrasion

Wood et al. defined abrasion as a phenomenon that promotes pathological wear, through biomechanical friction processes on the tooth substance (7). Amaral et al. complemented that abrasion is a demineralization process of the tooth structure or restoration, without bacterial involvement, caused by harmful habits in a slow, gradual and progressive way (3). Haralur et al. argued that abrasion is mechanical wear caused by constant contact with objects or substances (1).

The morphology of abrasion injury presents from initial lesions with light horizontal scratches on the tooth surface, without the presence of plaque or calculus due to aggressive and constant mechanical removal, evolving to a more oval, saucer-like shape (2, 19, 20). Abrasion may be associated with the technique, force and frequency of brushing as important factors related to the development of NCCL. In addition, it is associated with the abrasiveness of dentifrices, coarse foods and harmful oral habits (1, 30). Another factor that may influence the abrasive process is the stiffness of the toothbrush filaments and the shape of their ends (1, 3). Therefore, brushing technique alone is not a factor considered important for the appearance of abrasive lesions (3).

The findings of Amaral et al. indicated that brushes with soft bristles could be less abrasive than brushes with stiffer ones. However, if they are accompanied by non-fluoridated acid dentifrices, they would cause pathological wear, as they would retain a greater amount of paste for longer (3). Thus, the fluoride content is extremely important because it promotes a low pH balance and protects the tooth structure against wear (3). Moreover, an interaction between fluoride pastes and brushing twice a day contributes to a 30% reduction in erosion (3).

Nevertheless, studies by Bizhang et al. reported that soft bristle brushes promote greater dentin loss than stiff bristle brushes (31). Dyer et al. suggested that soft bristle brushes could lead to increased abrasion due to greater incorporation of toothpaste (32). Accordingly, Litonjua et al. argued that toothpaste has greater significance regarding tooth structure loss as the toothbrush solely plays the role of toothpaste carrier (33). However, Haralur et al. pointed out that patients with NCCL used the stiff-bristled toothbrush quite often along with the horizontal brushing technique (1). In addition, it was found that the abrasive phenomenon occurs more exacerbated in linear than in rotary brushing and that, added to the frequency, will result in a considerable increase in pathological wear (3).

The gingival retraction does not have a direct relationship with the trauma caused by aggressive brushing (3). However, when tooth wear is provoked, as a consequence there are: decrease in cemento, epithelial adhesion and loss of alveolar bone, inducing the gingival retraction. (3, 17).

The studies by Bartlett DW and Shah P indicated that there is little evidence suggesting that wear is caused solely by abrasion (20). Pathological wear is only achieved when a combination of abusive brushing and the predominant chemical erosion process occurs (3). The healthy tooth structure is weakened after a biocorrosive process, becoming less resistant to other forms of wear, such as abrasion (9). Biocorrosion and abrasion are synergistically and additively related, contributing to both enamel and dentin wear (3).

Adequate oral hygiene prevents periodontal and carious pathologies, while hygiene based on frequent aggressive brushing has the potential to cause pathological wear, especially if performed immediately after meals rich in acidic substances, after the ingestion of carbonated beverages or if any of these are retained for a prolonged time in the oral cavity (1, 3). These factors together with toothpastes are capable of triggering gingival retraction, enamel loss, exposure of cervical dentin, and opening of the tubular system, consequently provoking dentin hypersensitivity and abrasive phenomenon (3)

5. Relationship between dentin hypersensitivity and NCCL

As NCCL tend to expose the dentin, which generates considerable problems such as dentin

hypersensitivity (9, 34), its understanding arouses the interest of dental surgeons. This clinical condition is the main symptom of the initial process of wear of the NCCL and is defined as an acute and short pain, caused when the dentin is exposed with its network of dentinal tubules open in front of thermal, evaporative, tactile, osmotic and chemical stimuli (3, 5). The etiological factors of NCCL contribute to the onset and development of dentin hypersensitivity and it is of utmost importance to control them as an initial part of the treatment (9, 34).

CONCLUSIONS

In this study we observed how complex tracking the origin and development of NCCL is. We conclude that although much of the recent research considers that the disease has a multifactorial cause, there are still controversies involving its etiology. Thus, further work and systematic literature reviews are needed to better elucidate the onset and development of NCCL. Understanding the etiology of the disease is an indispensable step to properly plan its treatment, besides contributing to prevent of future lesions and stagnate existing ones.

The authors declare no conflict of interest.

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